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NEWS 24	AUG 15	CAplus currency for Korean patents enhanced
NEWS 25	AUG 25	CA/CAplus, CASREACT, and IFI and USPAT databases enhanced for more flexible patent number searching
NEWS 26	AUG 27	CAS definition of basic patents expanded to ensure comprehensive access to substance and sequence information

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=> S Bakker AND OX40-receptor
L1            0 BAKKER AND OX40-RECEPTOR

=> S Bakker AND OX40
L2            0 BAKKER AND OX40

=> S Antibody (S) OX40 (S) Receptor AND pd<=20041213
   2 FILES SEARCHED...
L3           29 ANTIBODY (S) OX40 (S) RECEPTOR AND PD<=20041213

=> Dup Rem L3
PROCESSING COMPLETED FOR L3
L4            12 DUP REM L3 (17 DUPLICATES REMOVED)
                 ANSWERS '1-6' FROM FILE MEDLINE
                 ANSWERS '7-12' FROM FILE CAPLUS

=> D Ti l4 1-12

L4   ANSWER 1 OF 12     MEDLINE on STN                  DUPLICATE 1
TI   Immunotherapy with OX40L-Fc or anti-CTLA-4 enhances local tissue responses
     and killing of Leishmania donovani.

L4   ANSWER 2 OF 12     MEDLINE on STN                  DUPLICATE 2
TI   Signaling through OX40 (CD134) breaks peripheral T-cell tolerance.

L4   ANSWER 3 OF 12     MEDLINE on STN                  DUPLICATE 3
TI   Critical contribution of OX40 ligand to T helper cell type 2
     differentiation in experimental leishmaniasis.
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L4 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 4
TI Expression of OX40 and OX40 ligand (gp34) in the normal and myasthenic thymus.

L4 ANSWER 5 OF 12 MEDLINE on STN DUPLICATE 5
TI OX40 is differentially expressed on activated rat and mouse T cells and is the sole receptor for the OX40 ligand.

L4 ANSWER 6 OF 12 MEDLINE on STN DUPLICATE 6
TI Overcoming tumor necrosis factor and drug resistance of human tumor cell lines by combination treatment with anti-Fas antibody and drugs or toxins.

L4 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Vaccines comprising TLR agonist, TNF/TNF receptor agonist and antigen for inducing cellular immune response against infection or tumor

L4 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Fusion proteins of cytokines and cell surface proteins for cell or tissue-specific activation of TNF receptors without the use of antibodies

L4 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Agonistic anti-human OX40 receptor scFv antibodies for enhancing antigen-specific immune response

L4 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method for enhancing an antigen specific immune response with OX-40 ligand

L4 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI Augmentation versus inhibition: effects of conjunctional OX-40 receptor monoclonal antibody and IL-2 treatment on adoptive immunotherapy of advanced tumor

L4 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
TI OX40/OX40 ligand system and immunoregulation

=> D Ibib abs L4 1-12

L4 ANSWER 1 OF 12 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004215902 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15114677
TITLE: Immunotherapy with OX40L-Fc or anti-CTLA-4 enhances local tissue responses and killing of Leishmania donovani.
AUTHOR: Zubairi Soombul; Sanos Stephanie L; Hill Sue; Kaye Paul M
CORPORATE SOURCE: Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, London, GB.
SOURCE: European journal of immunology, (2004 May) Vol. 34, No. 5, pp. 1433-40.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 29 Apr 2004
Last Updated on STN: 30 Jun 2004
Entered Medline: 29 Jun 2004
AB Enhancing granuloma development and effector function, but without inducing the pathology associated with excess granulomatous inflammation, poses a major challenge for immunotherapeutic intervention against

diseases such as visceral leishmaniasis (VL). Here, we demonstrate that a chimeric fusion protein (OX40L-Fc) which stimulates T cells through OX40 and a monoclonal antibody which blocks CTLA-4, an inhibitory receptor on T cells, both enhanced the rate of granuloma maturation, CD4(+) T cell proliferation, and killing of Leishmania. Costimulation-based therapy induced no adverse fibrotic or necrotic reactions, and had no significant effect on the levels of endogenous anti-inflammatory cytokines (IL-10 and TGF-beta). Furthermore, both OX40L-Fc and anti-CTLA4 could be co-administered with conventional anti-leishmanial drugs. Until now, enhancing T cell immunity by the manipulation of costimulatory pathways has only received serious attention for cancer immunotherapy, but our data provide a compelling argument for the evaluation of this approach in human VL and other infectious diseases.

L4 ANSWER 2 OF 12 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001432003 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11479622
TITLE: Signaling through OX40 (CD134) breaks peripheral T-cell tolerance.
AUTHOR: Bansal-Pakala P; Jember A G; Croft M
CORPORATE SOURCE: Division of Immunochemistry, La Jolla Institute for Allergy and Immunology, San Diego, California, USA.
CONTRACT NUMBER: AI42944 (United States NIAID)
SOURCE: Nature medicine, (2001 Aug) Vol. 7, No. 8, pp. 907-12.
Journal code: 9502015. ISSN: 1078-8956.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 27 Aug 2001
Last Updated on STN: 27 Aug 2001
Entered Medline: 23 Aug 2001
AB Peripheral T-cell tolerance is a mechanism to limit autoimmunity, but represents a major obstacle in diseases such as cancer. Tolerance is due to limited accumulation of antigen-specific T cells accompanied by functional hypo-responsiveness, and is induced by antigen encounter in a non-inflammatory environment. In contrast to advances in preventing induction of T-cell tolerance, there has been little progress in defining targets to reverse established tolerance. Here we show that signals from a single dose of an agonistic antibody against OX40 (CD134, a member of the tumor necrosis-factor family of receptors) can break an existing state of tolerance in the CD4+ T-cell compartment. OX40 signals promote T-cell expansion after the hypo-responsive phenotype is induced and restore normal functionality. These data highlight the potent costimulatory capacity of OX40, and indicate OX40 as a target for therapeutic intervention in a variety of related diseases.

L4 ANSWER 3 OF 12 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000105361 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10637281
TITLE: Critical contribution of OX40 ligand to T helper cell type 2 differentiation in experimental leishmaniasis.
AUTHOR: Akiba H; Miyahira Y; Atsuta M; Takeda K; Nohara C; Futagawa T; Matsuda H; Aoki T; Yagita H; Okumura K
CORPORATE SOURCE: Department of Immunology, Juntendo University School of Medicine, Tokyo 113-8421, Japan.
SOURCE: The Journal of experimental medicine, (2000 Jan 17) Vol. 191, No. 2, pp. 375-80.

Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 9 Mar 2000
Last Updated on STN: 9 Mar 2000
Entered Medline: 22 Feb 2000

AB Infection of inbred mouse strains with Leishmania major is a well characterized model for analysis of T helper (Th)1 and Th2 cell development in vivo. In this study, to address the role of costimulatory molecules CD27, CD30, 4-1BB, and OX40, which belong to the tumor necrosis factor receptor superfamily, in the development of Th1 and Th2 cells in vivo, we administered monoclonal antibody (mAb) against their ligands, CD70, CD30 ligand (L), 4-1BBL, and OX40L, to mice infected with L. major. Whereas anti-CD70, anti-CD30L, and anti-4-1BBL mAb exhibited no effect in either susceptible BALB/c or resistant C57BL/6 mice, the administration of anti-OX40L mAb abrogated progressive disease in BALB/c mice. Flow cytometric analysis indicated that OX40 was expressed on CD4(+) T cells and OX40L was expressed on CD11c(+) dendritic cells in the popliteal lymph nodes of L. major-infected BALB/c mice. In vitro stimulation of these CD4(+) T cells showed that anti-OX40L mAb treatment resulted in substantially reduced production of Th2 cytokines. Moreover, this change in cytokine levels was associated with reduced levels of anti-L. major immunoglobulin (Ig)G1 and serum IgE. These results indicate that anti-OX40L mAb abrogated progressive leishmaniasis in BALB/c mice by suppressing the development of Th2 responses, substantiating a critical role of OX40-OX40L interaction in Th2 development in vivo.

L4 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2001302716 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11071109
TITLE: Expression of OX40 and OX40 ligand (gp34) in the normal and myasthenic thymus.
AUTHOR: Onodera J; Nagata T; Fujihara K; Ohuchi M; Ishii N; Sugamura K; Itoyama Y
CORPORATE SOURCE: Department of Neurology, Tohoku University School of Medicine, Sendai, Japan.
SOURCE: Acta neurologica Scandinavica, (2000 Oct) Vol. 102, No. 4, pp. 236-43.
Journal code: 0370336. ISSN: 0001-6314.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 4 Jun 2001
Last Updated on STN: 4 Jun 2001
Entered Medline: 31 May 2001

AB OBJECTIVES: To examine the expression of OX40, an activated memory T-cell marker, and its ligand (OX40L), a set of molecules for T-cell-B-cell interaction, and other lymphocyte activation markers in the thymuses of myasthenia gravis (MG) and controls. MATERIAL AND METHODS: We studied the expression of OX40, OX40L, IL-2Ralpha and HLA-DR in the thymic tissues of MG and controls using immunocytochemistry and flowcytometry. RESULTS: In both hyperplastic thymus of MG and control thymus, OX40+ cells were scattered mainly in the medulla with much fewer OX40L+ cells being distributed in the corticomedullary junctions. IL-2Ralpha and HLA-DR were

expressed in the medulla at higher frequencies as compared with OX40 in controls as well as MG. In contrast, the numbers of OX40+ cells around the germinal centers (GC) were significantly greater than those of control thymuses, and some mononuclear cells in GC were OX40L+. A considerable number of OX40+ cells were seen in the thymic tissues adjacent to thymomas. OX40+ cells were CD4+ CD8- or CD4+ CD8+ and were mostly HLA-DR-. (The coexpression of OX40 and IL-2Ralpha on activated CD4+ T cells was previously reported.) CONCLUSION: OX40 , expressed in a fraction of activated CD4+ T cells, may be upregulated in thymic tissues adjacent to GC and thymoma in MG, and OX40 may interact with OX40L in GC to enhance anti-acetylcholine receptor antibody production in MG.

L4 ANSWER 5 OF 12 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 1996350485 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8765008
TITLE: OX40 is differentially expressed on activated rat and mouse T cells and is the sole receptor for the OX40 ligand.
AUTHOR: al-Shamkhani A; Birkeland M L; Puklavec M; Brown M H; James W; Barclay A N
CORPORATE SOURCE: MRC Cellular Immunology Unit, University of Oxford, GB.
SOURCE: European journal of immunology, (1996 Aug) Vol. 26, No. 8, pp. 1695-9.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 6 Nov 1996
Last Updated on STN: 6 Nov 1996
Entered Medline: 24 Oct 1996
AB OX40, a member of the tumor necrosis factor (TNF) receptor/nerve growth factor (NGF) receptor superfamily was first identified as a marker of activated rat CD4+ cells with the MRC OX40 monoclonal antibody (mAb). A ligand for OX40 (called OX40 ligand or OX40L) has recently been identified and has sequence similarity to TNF. Mouse OX40L-immunoglobulin fusion protein (OX40L-Ig) binds to activated mouse CD4+ and CD8+ cells (Baum, P. R. et al., EMBO J. 1994. 13: 3992) suggesting that OX40 could have a differential pattern of expression on mouse and rat T cells. This, however, did not rule out the presence of an alternative receptor on CD8+ cells that also binds the OX40L. We have compared the binding of the MRC OX40 mAb with that of OX40L-Ig to activated rat lymph node cells and show that both recognize the same protein, namely OX40 which is expressed on CD4+ and CD4+ CD8 alpha+ cells, but not on CD4-CD8+ cells. We have raised a new mAb (MRC OX86) using recombinant mouse OX40 protein and show by two-color flow cytometry that mouse OX40 is expressed on CD4 and CD8 single-positive cells. In addition, the new MRC OX86 mAb, unlike the MRC OX40 mAb, did not block binding of the OX40L. We conclude that OX40 is differentially expressed on activated mouse and rat T cells and is the sole receptor for the OX40L.

L4 ANSWER 6 OF 12 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 1993265460 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7684321
TITLE: Overcoming tumor necrosis factor and drug resistance of human tumor cell lines by combination treatment with anti-Fas antibody and drugs or toxins.
AUTHOR: Morimoto H; Yonehara S; Bonavida B
CORPORATE SOURCE: Department of Microbiology and Immunology, UCLA School of

Medicine, University of California 90024.
SOURCE: Cancer research, (1993 Jun 1) Vol. 53, No. 11, pp. 2591-6.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 2 Jul 1993
Last Updated on STN: 3 Feb 1997
Entered Medline: 21 Jun 1993

AB Monoclonal mouse anti-Fas antibody is directed against Fas antigen, a M(r) 36,000 encoded polypeptide that belongs to the family of cell surface proteins which includes nerve growth factor receptor , tumor necrosis factor (TNF) receptors, B-cell antigen CD40, and T-cell antigens OX40. Anti-Fas antibody mimics TNF-alpha in its cytolytic activity but not in other TNF-alpha-mediated activities. Thus, we examined if anti-Fas antibody synergizes in cytotoxicity with toxins and drugs. The present studies demonstrate that anti-Fas antibody in combination with diphtheria toxin (DTX), Adriamycin, or cis-platinum results in enhanced cytotoxicity and synergy and also overrides resistance to TNF, drugs, or toxins when tested against a battery of human tumor cell lines. Synergy with anti-Fas and DTX requires that DTX is enzymatically active, since inhibitors of DTX-mediated protein synthesis inhibition resulted in loss of synergy. When the plant toxin ricin was used, there was no synergy with anti-Fas antibody but rather an additive effect. The synergy was not obtained in a TNF receptor-negative line but was achieved with other anti-Fas-resistant lines. Cell lines resistant to either Adriamycin or cis-platinum were rendered sensitive by the combination of drug and anti-Fas antibody. Further, combination treatment of anti-Fas and Adriamycin overcame resistance of the gp 170-expressing, multidrug-resistant MDR ovarian line. In all cases, cytotoxicity was augmented by pretreatment of target cells with gamma-interferon which upregulates Fas antigen expression. These results show that anti-Fas antibody can synergize in cytotoxicity with toxins and chemotherapeutic drugs, and combination treatment can reverse resistance to TNF, toxins, and/or drugs.

L4 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:589386 CAPLUS
DOCUMENT NUMBER: 141:139130
TITLE: Vaccines comprising TLR agonist, TNF/TNF receptor agonist and antigen for inducing cellular immune response against infection or tumor
INVENTOR(S): Noelle, Randolph J.; Ahonen, Cory L.; Kedl, Ross M.
PATENT ASSIGNEE(S): 3M Innovative Properties Company, USA
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004060319	A2	20040722	WO 2003-US41796	20031230 <--
WO 2004060319	A3	20041104		

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CA 2511538	A1	20040722	CA 2003-2511538	20031230 <--
US 20040141950	A1	20040722	US 2003-748010	20031230 <--
US 7387271	B2	20080617		
AU 2003300184	A1	20040729	AU 2003-300184	20031230 <--
EP 1578419	A2	20050928	EP 2003-800433	20031230

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IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2006512391	T	20060413	JP 2004-564947	20031230
PRIORITY APPLN. INFO.:			US 2002-437398P	P 20021230
			WO 2003-US41796	W 20031230

AB The present invention provides immunostimulatory combinations. Generally, the immunostimulatory combinations include a TLR agonist, a TNF or TNF receptor agonist and an tumor antigen or viral, bacterial or parasitic antigen. The TLR agonist is an agonist of TLR1-10 e.g. IRM compound, MALP-2, LPS, polyIC, CpG or any combination. The TNF agonist is an agonist or antibody against CD40L, OX40 ligand, 4-1BB ligand, CD27, CD30 ligand, TNF-α, TNF-β, RANK ligand, LT-α, LT-β, GITR ligand or LIGHT. The TNF receptor agonist is an antibody or agonist of CD40, OX40, 4-1BB, CD27 ligand, CD30, TNFR2, RANK, LT-αR, LT-βR, HVEM, GITR, TROY or RELT. These immunostimulatory combinations are useful for inducing Th1 immune response or antigen-specific CD8+ effector and memory T cell response against infectious and neoplastic conditions.

L4 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:328886 CAPLUS
DOCUMENT NUMBER: 140:337940
TITLE: Fusion proteins of cytokines and cell surface proteins for cell or tissue-specific activation of TNF receptors without the use of antibodies
INVENTOR(S): Pfizenmaier, Klaus; Wajant, Harald
PATENT ASSIGNEE(S): Germany
SOURCE: Ger. Offen., 16 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10247755	A1	20040422	DE 2002-10247755	20021014 <--
DE 10247755	B4	20060119		
WO 2004035794	A1	20040429	WO 2003-EP11357	20031014 <--

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AU 2003271721	A1	20040504	AU 2003-271721	20031014 <--

EP 1551977 A1 20050713 EP 2003-753550 20031014
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2006502720 T 20060126 JP 2004-544215 20031014
US 20050244370 A1 20051103 US 2005-105172 20050413
PRIORITY APPLN. INFO.: DE 2002-10247755 A 20021014
WO 2003-EP11357 W 20031014

AB Fusion proteins of weakly active or inactive derivs. of tumor necrosis factors and cell surface proteins are described for use in cell-specific activation of tumor necrosis factor receptors. The tumor necrosis factor effector domain is a fragment, in particular the extracellular domains, of a member of the TNF ligand family with little or no biol. activity. The cell surface mol. bonding domain is derived from a cell type-specific membrane protein that is not derived from an Ig. Furthermore the invention makes coding nucleic acid constructs available for polypeptides, containing these vectors, thereby transfixed host cells, for the polypeptides the named inventive subject containing pharmaceutical compns., procedures for the production of the inventive polypeptides as well as uses of inventive objects for therapeutic purposes. Fusion proteins of CD40 antigen and Fas ligand or TRAIL are described.

L4 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:1007016 CAPLUS
DOCUMENT NUMBER: 140:58439
TITLE: Agonistic anti-human OX40 receptor scFv antibodies for enhancing antigen-specific immune response
INVENTOR(S): Bakker, Alexander Berthold Hendrik; Meester-Rood, Pauline Marie Louise; Bakker, Adrianus Quirinus
PATENT ASSIGNEE(S): Crucell Holland, B.V., Neth.
SOURCE: PCT Int. Appl., 158 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003106498	A2	20031224	WO 2003-EP6341	20030613 <--
WO 2003106498	A3	20040401		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2489004	A1	20031224	CA 2003-2489004	20030613 <--
AU 2003257419	A1	20031231	AU 2003-257419	20030613 <--
EP 1525223	A2	20050427	EP 2003-759966	20030613
EP 1525223	B1	20071121		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
NZ 536746	A	20070223	NZ 2003-536746	20030613
ES 2295639	T3	20080416	ES 2003-759966	20030613
US 20060281072	A1	20061214	US 2004-517941	20041213
PRIORITY APPLN. INFO.:			WO 2002-NL389 A	20020613
			WO 2003-EP6341 W	20030613

AB The invention provides binding mols., such as human binding mols., that bind to and stimulate the human 0X40-receptor. The invention also provides nucleic acids encoding such binding mols. Methods for producing and screening such binding mols. are also provided by the present invention. Said binding mols. and nucleic acids are useful in the stimulation of human T-cells, and can be used to enhance antigen-specific immune responses. Immunoconjugates comprising the binding mols. are useful for enhancing the immune response against tumor, viral or bacterial antigen and for treating tumor, viral and bacterial disease in human or animal.

L4 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:809019 CAPLUS
DOCUMENT NUMBER: 135:343303
TITLE: Method for enhancing an antigen specific immune response with OX-40 ligand
INVENTOR(S): Weinberg, Andrew D.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 30 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6312700	B1	20011106	US 1999-255363	19990223 <--
US 20020054873	A1	20020509	US 2001-946832	20010904 <--
US 20070207159	A1	20070906	US 2006-529956	20060929

PRIORITY APPLN. INFO.:
US 1998-75801P P 19980224
US 1999-255363 A3 19990223
US 2001-946832 A1 20010904

AB Provided are compns. and methods for enhancing the immune response of a mammal to an antigen by engaging the OX-40 receptor on the surface of T-cells are disclosed, comprising administering to the mammal a composition comprising a purified OX-40 receptor binding agent and a pharmaceutically acceptable carrier, wherein said composition is administered to the mammal such that the OX-40 receptor binding agent is presented to T-cells of the mammal during or shortly after priming of the T-cells by the antigen. Such compns. and methods can be used in immunization and cancer treatment.
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:884232 CAPLUS
DOCUMENT NUMBER: 136:149820
TITLE: Augmentation versus inhibition: effects of conjunctional OX-40 receptor monoclonal antibody and IL-2 treatment on adoptive immunotherapy of advanced tumor
AUTHOR(S): Kjaergaard, Jorgen; Peng, Liaomin; Cohen, Peter A.; Drazba, Judith A.; Weinberg, Andrew D.; Shu, Suyu
CORPORATE SOURCE: Center for Surgery Research, Cleveland Clinic Foundation, Cleveland, OH, 44195, USA
SOURCE: Journal of Immunology (2001), 167(11), 6669-6677
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Therapeutic efficacy of adoptive immunotherapy of malignancies is

proportional to the number of effector T cells transferred. Traditionally, exogenous IL-2 treatment has been used to promote the survival and function of transferred cells. Recently, we described the therapeutic effects of in vivo ligation of the costimulatory receptor, OX-40R, on activated T cells during early tumor growth. In this study, we examined the effects of IL-2 and OX-40R mAb on adoptive immunotherapy of advanced tumors. For treatment of 10-day 3-methylcholanthrene 205 pulmonary metastases, systemic transfer of 50 + 106 activated tumor-draining lymph node T cells resulted in >99% reduction of metastatic nodules. With either IL-2 or OX-40R mAb conjunctional treatment, only 20 + 106 cells were required. Advanced 10-day 3-methylcholanthrene 205 intracranial tumors could be cured by the transfer of 15 + 106 L-selectinlow T cells derived from draining lymph nodes. In this situation, IL-2 administration inhibited therapeutic effects of the transferred cells. By contrast, 5 + 106 T cells were sufficient to cure all mice if OX-40R mAb was administrated. Studies on trafficking of systemically transferred T cells revealed that IL-2, but not OX-40R mAb, impeded tumor infiltration by T cells. Tumor regression required participation of both CD4 and CD8 T cells. Because only CD4 T cells expressed OX-40R at cell transfer, direct CD4 T cell activation is possible. Alternatively, OX-40R might be up-regulated on transferred T cells at the tumor site, rendering them reactive to the mAb. Our study suggests OX-40R mAb to be a reagent of choice to augment T cell adoptive immunotherapy in clin. trials.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2000:907527 CAPLUS
DOCUMENT NUMBER: 134:16279
TITLE: OX40/OX40 ligand system and immunoregulation
AUTHOR(S): Sugamura, Kazuo
CORPORATE SOURCE: Sch. Med., Tohoku Univ., Japan
SOURCE: Nippon Hifuka Gakkai Zasshi (2000), 110(12, Rinjizokango), 1811-1812
CODEN: NHKZAD; ISSN: 0021-499X
PUBLISHER: Nippon Hifuka Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review, with 6 refs., on expression of OX40 ligand (OX40L), a ligand of OX40 (CD134), a member of TNF receptor family, on CD40-activated dendritic cells and B cells stimulated with CD40 and anti-IgM antibody, dysfunction of antigen-presenting cells (APC) in OX40L-deficient mice, and possible involvement of OX40 /OX40L system in onset of autoimmune diseases.

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SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 21:23:42 ON 08 SEP 2008